Year:	2004	2005	2006	2007	2008	2009	P value
Mean age, years	63	64	64	64	65	64	0.0052
Prior myocardial infarction, %	14.0	13.8	12.3	10.3	9.4	9.7	<0.0001
Killip 4 on admission, %	9.8	7.7	6.4	5.7	5.2	5.3	<0,0001
Clopidogrel, %	57.1	66.6	72.1	85.1	94.0	96.9	<0.0001
Beta-blocker, %	73.5	71.5	67.4	71.9	73.2	76.2	<0.0001
Statin, %	72.0	74.0	72.0	76.3	78.5	80.9	<0.0001
ACE inhibitor, %	65.9	67.6	65.7	69.6	70.3	71.1	<0.0001
Reperfusion therapy, %	64.5	63.2	62.8	67.9	73.2	81.1	<0.0001
Thrombolysis, %	11.2	9.1	6.3	3.4	1.9	1.2	<0.0001
Primary angioplasty (pPCI), %	53.2	54.1	56.5	64.4	71.3	79.8	<0.0001
Bypass surgery (CABG), %	7.6	7.7	6.0	6.3	6.4	5.6	<0.0001
Median time to discharge, days	6	6	5	5	4	5	<0.0001
In-hospital major bleeding, %	0.9	0.7	0.7	1.2	2.2	1.9	<0.0001
In-hospital re-infarction, %	3.1	4.6	2.9	3.4	2.6	1.7	<0.0001
In-hospital mortality, %	10.0	8.9	7.5	6.4	6.2	6.3	<0.0001
30-day mortality, %	12.6	11.8	10.7	9.8	9.6	9.6	<0.0001
12-month mortality, %	17.9	17.6	15.9	15.4	15.4	14.7	<0.0001

Conclusion: Increase in adherence to the guidelines leads to decrease in short and mid-term mortality in STEMI, but the increase in major bleeding complications was noted.

TCT-349

Monocyte Platelet Aggregate act as Platelet Function Assay and Prognostic Marker for Acute Myocardial Infarction

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Background: During acute myocardial infarction (AMI), degranulated platelets adhere to the monocytes and form monocyte platelet aggregate (MPA). The level of MPA can therefore reflect the pathological change and indicate the degree of inflammation and thrombosis in AMI. Recently, MPA assay has been developed for in vivo platelet function assessment. MPA may act as a biomarker for ACS.
Methods: In this single-center prospective observational study, we recruited 29 consecutive AMI patients and 19 healthy controls. Blood samples in first 24 hrs of AMI and Day 5 after dual antiplatelet treatment were taken from patients. MPA level was measured by flow cytometry. MPA in circulating blood were displayed in a dual-fluorescence dot plot of CD14-FITC and CD41-PE. MPA level of the health subjects and AMI patients were compared. In wave one analysis, patients were grouped into high MPA group and low MPA group by upper limit of the normal subjects, in which baseline characteristics and clinical endpoints between two groups were compared. In wave two analysis, patients with rising trend of MPA after dual antiplatelet treatment and patients with decreasing trend of MPA were compared. The endpoint is composite cardiovascular endpoints of death, recurrent ACS, cardiogenic shock, heart failure and ventricular tachycardia/ventricular fibrillation (VT/VF) in post AMI day 30.
Results: MPA level in AMI patients (mean= 33) are significantly higher than the normal health subjects (mean=25). ($p<0.05$). In wave one analysis, high MPA group (n=9) and low MPA (n=20) group reaching the endpoint were 55% (n=5) and 30% (n=6) respectively. (p=0.2). In wave two analysis, rising MPA group (n= 12) showed 58.3 % (n=7) patients reaching endpoint. Decreasing trend MPA group (n=9) showed 11.1% (n=1) patient reaching endpoint. Rising MPA group showed a trend of more post MI complications compared to decreasing MPA group but statistically, it is not significant. (p= 0.06).
Conclusion: MPA significantly elevated in AMI patients. There is a trend that high MPA and rising MPA in AMI patients will have more post MI complications.

TCT-350

Very Late Hazard with Primary Stenting versus Primary PTCA for STEMI: A 16 Year Single Center Experience

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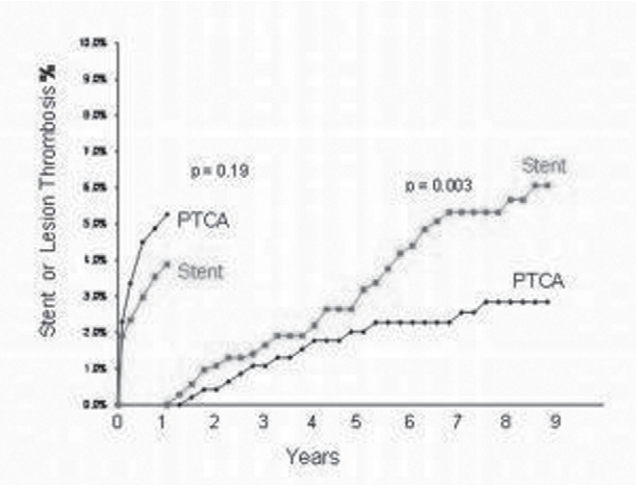
Background: Stenting compared with PTCA for STEMI reduces TVR and target vessel re-occlusion at 6-12 months. Comparison of outcomes beyond 12 months has not been studied. We hypothesized that there may be a very late hazard with stenting vs PTCA due to very late stent thrombosis (ST).
Methods: From 1995-2010 consecutive pts with STEMI were treated with primary PTCA (n=601) or primary stenting with BMS or DES (n=1594). Clinical and angiographic follow-up were obtained at 1-16 yrs. Comparable definitions were used for ST (ARC definite or probable) and lesion thrombosis (LT).
Results: Pts treated with PTCA were older, more often female, had more hypertension, more 3V CAD, smaller vessels, more TIMI 0-1 flow at initial angiography, and longer reperfusion times. At 1 yr there were mild trends for lower mortality, MI and ST/LT with stenting vs PTCA. In landmark analyses after 1 yr, pts treated with stenting vs PTCA had more very late ST/LT (Figure) and more TV MI but no differences in death or total MI. Most of the differences in ST/LT and TV MI between stents and PTCA were due to differences between DES and PTCA with smaller differences between BMS and PTCA. After adjusting for baseline risk, stents vs PTCA had more very late ST/LT (HR 2.63, 95% CI 1.40-4.95, p=0.003) and more very late TV MI (HR 2.98, 95% CI 1.68-5.31, p<0.001).



Conclusion: There appears to be a very late hazard with primary stenting compared with primary PTCA for STEMI with more very late ST/LT and more TV MI. These data should encourage new strategies for prevention of very late ST with both BMS and DES including the development of bio-absorbable stents and polymers.

TCT-351

Impact of Bleeding on Mortality in Patients Referred for Primary PCI

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Background: There is limited contemporary data on factors that predict bleeding in the setting of primary PCI, and on the impact of bleeding on mortality.
Methods: We identified pts referred for primary PCI from our database. Standard therapy before catheterization included ASA 160mg, clopidogrel 600mg and UFH 60 u/kg (max 4000). Bleeding was defined as TIMI major/minor bleeding. We used logistic regression to determine baseline variables associated with bleeding during the initial hospitalization and all-cause mortality at 180 days.
Results: Between May 2005 and July 2010, 2032 consecutive pts with STEMI were referred for primary PCI. Primary PCI was performed in 93% of pts and transradial access was used in 12%. Bivalirudin was used in to 29% of pts and glycoprotein 2b/3a inhibitors to 36%. Bypass surgery was performed in 4%. TIMI major/minor bleeding occurred in 14.8% of pts: TIMI major in 4.5% and TIMI minor in 10.3%. Blood transfusion was needed in 6.3% of pts. Independent predictors of bleeding were Killip class, anterior STEMI, transfemoral access, age, anemia, serum creatinine, door-to-balloon time, height and the use of glycoprotein 2b/3a vs. bivalirudin. Mortality at 180 days was 7.7% for the entire group: 18.5% in pts with bleeding vs. 5.6% in pts without bleeding, $p<0.0001$. The mortality rate was 24.4% in pts with major bleeding and 15.8% in pts with minor bleeding. The results of the multivariable analysis to determine predictors of mortality at 180 days are shown in the Table below.

Variable	Odds Ratio	LCL	UCL	p-value
TIMI Minor Bleed	3.71	2.09	6.59	<0.0001
TIMI Major Bleed	4.47	2.21	9.03	<0.0001
Diabetes Mellitus	2.97	1.74	5.01	<0.0001
Systolic Blood Pressure	0.99	0.98	0.99	0.02
Heart Rate	1.01	1.004	1.023	0.0007
Killip Class	2.02	1.58	2.58	<0.0001
Age	1.06	1.04	1.08	<0.0001

Conclusion: In the real world setting, TIMI major and TIMI minor bleeding are important predictors of mortality in pts referred for primary PCI. Altering factors that predict bleeding could lead better clinical outcomes.

TCT-352

Prior Coronary Artery Bypass Graft (CABG) Patients Treated with Primary Percutaneous Coronary Intervention (PPCI) Have Higher Long-Term Major Adverse Cardiovascular Event (MACE) Rates

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Background: Limited information exists regarding procedural success and clinical outcomes of patients with prior CABG undergoing PPCI for STEMI. We sought to compare outcomes in this group.
Methods: Data was analysed from a prospective database on 2322 STEMI patients undergoing PPCI between 2004-2010 at a London centre. 104 of 2,322 (4.5%) patients had prior CABG. Information was entered at the time of procedure and outcome assessed by all-cause mortality information.

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